REPLY

I was pleased that Alstadt and Eaglstein found the recent article by Takashima and myself to be of interest and have answered their questions below.

A. We have not tested carefully whether fibronectin plays a role in the initiation of keratinocyte migration out of explants. It has been our experience, however, that only about 25% of explants set up with fibronectin-depleted serum exhibit migration of cells from all around the explant compared with 60–70% of the explants cultured in fibronectin-containing medium.

B. The level of antifibronectin antibodies was chosen based upon preliminary studies in which this concentration was found to inhibit keratinocyte attachment and spreading on fibronectin-coated substrata. This antibody is an IgG preparation but not affinity purified, and we do not know the ratio of antibody molecules to fibronectin molecules. Such antibody levels do not seem excessive since similar concentrations also have been shown to be required to inhibit adhesion of fibroblasts to fibronectin [1], and nonimmune IgG at similar or higher concentrations had no effect.

C. We have not studied the effect of fibronectin on epidermal cell mitosis, but Gilchrest et al demonstrated that fibronectin markedly increased keratinocyte plating efficiency [2].

D. Fibronectin receptor function appeared on day 2 as measured by the bead binding assay, which we have shown previously to be a more sensitive measure of receptors than cell attachment and spreading [3].

Finally, I should mention that the onset of fibronectin receptor function during wound healing in vivo has been described in a paper by Takashima, Billingham, and myself [4].

REFERENCES

1. Grinnell F, Feld MK: Initial adhesion of human fibroblasts in serum-free medium: possible role of secreted fibronectin. Cell 17:117–129, 1979
2. Gilchrest BA, Binding and phagocytosis of fibronectin-coated beads by BHK cells: receptor specificity and dynamics. J Cell Physiol 112:197–206, 1982
3. McAbee DD, Grinnell F: Calhoun JK, Maciceg T: Attachment and growth of human Keratinocytes in a serum-free environment. J Cell Physiol 124:240–246, 1985
4. Takashima A, Billingham RE, Grinnell F: Activation of rabbit keratinocyte fibronectin receptor function in vivo during wound healing. J Invest Dermatol 86:585–590, 1986

Frederick Grinnell, Ph.D.
The University of Texas
Health Science Center at Dallas
Dallas, Texas

Cytokine from Basal Cell Carcinomas Stimulates Collagenase Production

To the Editor:

The article "Stimulation of Skin Fibroblast Collagenase Production by a Cytokine Derived from Basal Cell Carcinomas" by Goslen, Eisen, and Bauer (85:161–164, 1985) has provoked great interest on this Continent where these cancers are extraordinarily common. We would like to congratulate the authors on their findings and to add in a small way, as yet unpublished, the statistical fact that in the superficial cicatrising basal cell carcinoma (BCC) there is much less stimulation of fibroblast collagenase. We have not used as sophisticated methods as the authors but will do so and expect to confirm this.

It is expected with respect to the superficial cicatrising BCC, that whilst inner areas of fibroblasts are not stimulated by cytokines, some of those at the edge are influenced by external cytokines and hence the notorious incidence of recurrence in this type of cancer, which is well known and often termed "field effect carcinogenesis." We would like to know if the authors used any superficial cicatrising BCCs for their source material and, second, if they have any comment to make about the fibroblasts accompanying the superficial fibrotic (cicatrising) BCC, since the morphoeic BCC is often confused with the latter in the literature. In our findings this has not been the case. In other words, the superficial BCC should not be histologically or biologically equalled with the morphoeic BCC which behaves much more in the fashion as described by authors Goslen, Eisen, and Bauer.

Malcolm Lane-Brown, B.Sc., M.B., B.S., F.A.C.D.
Sydney, N.S.W., Australia

Paul Forlot, Ph.D.
Paris, France

REPLY

We have read, with interest, the comments by Drs. Lane-Brown and Forlot regarding our recent publication in *The Journal of Investigative Dermatology* (May 1985, pp 161–164). Our study involved nodular basal cell carcinomas and did not involve any superficial fibrotic or morpheaform basal cell carcinomas. Therefore, we have no particular insights to the questions the authors raise in their letter. Obviously, it is our hope to investigate these tumors in the future as they do represent an intriguing aspect of epithelial cancer-matrix interacation.

J. Blake Goslen, M.D.
Arthur Z. Eisen, M.D.
Eugene A. Bauer, M.D.
Washington University Medical Center
St. Louis, Missouri